Supplementary reference

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Supplementary discussion:

One important risk factor that can predispose patients to the development of collapsing glomerulopathy is the presence of Apolipoprotein L1 (*APOL1*) risk alleles. The expression of APOL1 protein in podocytes will increase in response to type 1 interferons, leading to direct cellular injury during viral infection and other conditions that alter the activity of the immune system.[S3] The higher prevalence of *APOL1* risk alleles in African American patients could explain the higher prevalence of collapsing glomerulopathy seen in the setting of SARS-CoV2 infection. [S4] It is important to note, however, that collapsing glomerulopathy due to viral infection can occur without the presence of high-risk *APOL1* alleles.[S5] Prior to this case, our institution experienced 2 cases of collapsing FSGS associated with COVID-19 infection. One patient carried wild type *APOL1* alleles (G0/G0) and the other was homozygous for a high-risk variant (G1/G1).

The post-marketing studies of voriconazole investigated the risk of kidney injury in the patients with chronic kidney disease. One retrospective observational study examined 42 patients with mild kidney disfunction (CrCl <50 ml/min) receiving IV voriconazole. Thetypical dosing schedule of voriconazole consisted of a loading dose of 6 mg/kg followed by maintenance doses of 200 mg twice daily, which corresponds to a cumulative 71 g of SBECD over a 10-day course in a patient weighing 75 kg. The serum creatinine did not rise at the end of treatment.[S6] It is also noteworthy that SBECD is effectively dialyzable.

Additionally, though it is possible that remdesivir could have direct renal toxicity by inhibiting mammalian RNA polymerase, it is unlikely that our patient experienced direct toxicity by remdesivir, because its effect on mammalian RNA polymerase is only observed at very high dose and prolonged administration. Given the potential risks, patients with advanced CKD and AKI with eGFR cutoff either 50 or 30 ml/min/1.73 m^2 were excluded from the original trials comparing remdesivir with placebo. Therefore, an increased risk of kidney-related adverse events in the reported trials of remdesivir could have been missed.[S8]